# THE STATUS OF EXERCISE IN THE TUBERCULOUS CONSIDERED FROM A NEURO-MUSCULAR VIEWPOINT.

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It is the purpose of this paper to record the more important mechanistic changes incident with physiologic exercise, and at the same time to parallel the maladjustments as they occur in the individual who is suffering from an active tuberculosis.

Muscular exercise should be considered from the standpoint of a neuro-muscular system, and we should ever bear in mind the continual adjustments which are occurring in the respiratory and circulatory mechanism. Exercise produces a subconscious concentration of energies of the higher nerve centers, and it is this expenditure which we must conserve. Furthermore, an improper conception of its character will be attained if it is considered as a purely muscular act. For a person to enjoy muscular exercise it is necessary that the movements of the skeletal muscles and those of the respiratory and circulatory system should be coördinated and integrated into a unit, as they are indissolubly linked together.

Exercise is purely an outward expression of neurogenic action, and when once it is initiated, the different processes then occur in sequential order until the action resembles a machine; and considered from this viewpoint it is not difficult to understand that inefficiency of any part of the mechanism would inevitably spell defeat.

A man's nervous organization is as important as his muscular development, and his ability for performing muscular work is not altogether gauged by the size of the musculature. As a rule the heart is the limiting factor which gauges exercise. Every voluntary movement adds to the work of the heart: (*a*) by increasing the output because the larger venous inflow; (*b*) the blood is expelled against a higher arterial pressure.

The source of muscular energy is to a large extent derived from carbohydrates and fats, and the belief that protein furnishes a portion of energy has long been abandoned. The conversion of potential into kinetic energy involves the liberation of lactic acid, which is an intermediary product of carbohydrate combustion. During the process of muscular exercise lactic acid is oxidized within the muscle, if the exercise is not too violent; in event that it is strenuous it then escapes into the blood stream.

It might be well at this point to introduce the method we employ in all cases of active disease. Every case is immediately placed in bed, the period of rest is variable, and not infrequently we find it necessary to confine cases to the prone position from eighteen

months to two years. All trays are served in bed, and the only exercise permissible is toilet exercise. If there is one single factor which has enhanced the results in the last few years it has been the proper appreciation of rest. It is our plan to carry rest to the n'th power, and I am of the opinion that our results would be somewhat hastened if we insisted that the patient maintains the prone posture and not be allowed the laxity of sitting up in bed for long intervals.

From the foregoing remarks the influence of attitude may seem superfluous to the reader, but even a change of position from lying down to sitting or standing has a definite effect on both the consumption of oxygen and the pulse-rate. There is an approximate increase in the pulse-rate of 25 per cent and an increase in oxygen consumption of 20 per cent. This can be explained on the assumption that a greater number of impulses are coursing from the cerebral cortex to the bulb, or, in other words, utilizing the same mechanism brought into action as that occurring at the outset of exercise. Furthermore, very little energy is necessary when lying down, but if allowed to walk it requires 100 per cent more expenditure.

At the outset of exercise impulses pass from the higher centers, and probably mainly from the motor area of the cerebral cortex to the medulla. The irradiation of impulses which occur stimulates the vasomotor, respiratory and cardio-inhibitory center, raising blood-pressure and increasing pulse-rate and respiration. Simultaneous with this impulses are sent to the skeletal musculature via the pyramidal tracts and down the cord through the lateral splanchnic motor-cell columns to the V–XII thoracic segments, producing a constriction of the splanchnic area. With a contraction of the skeletal muscles and a forcing of the blood from the splanchnic area there is an increased venous pressure, and also the diastolic volume of the heart is greatly increased. When once exercise is thoroughly instituted the muscles squeeze the blood from the smaller capillaries into the larger veins and in this manner act as a subsidiary pump.

Coincident with muscular contraction lactic and carbonic acids are formed, also acid metabolites. It seems that in almost every type of exercise the oxygen supply is inadequate for complete oxidation, and particularly lactic acid makes its entrance into the blood stream, which immediately affects the hydrogen-ion concentration of the blood. There is an equilibrating force which now comes into action and the "buffer substances" or alkali reserve of the blood attempts to equalize the condition—that is, the lactic acid reacts with the plasma bicarbonate forming carbonic acid. Carbonic acid in solution acts as an acid, and this produces an increased pulmonary ventilation by its action upon the respiratory center. By increasing the pulmonary ventilation additional work is thrown upon the heart, as the output of the heart is directly proportional to the consumption of oxygen.

It is our observation in tuberculosis that a high-grade toxemia may exist without the presence of temperature. Toxemia in itself changes the chemical composition of the blood by raising hydrogen-ions, and this favors necrosis of tissue. Exercise in the individual actively tuberculous plus a moderate grade of toxemia can attain but one end, and that is tissue-destruction.

I am of the opinion that healing is greatly facilitated when the hydrogen-ion concentration of the blood remains as nearly as possible at a normal level. It is a notorious fact that a portion of the healing which occurs in tuberculosis is due to the deposition of lime salts; especially is this true if there have been areas of caseation. If some condition, such as exercise, is superimposed upon this preëxisting condition, then the reaction of the tissues swing toward the acid side, with a depletion of "buffer substances" in the blood. Hence the healing is delayed, as it is dependent in a measure upon the alkaline salts.

Bradley and Taylor[1] have put forward the view that the hydrogen-ion concentration within the tissues is an important factor in the building up or breaking down of tissues, as the case may be. They have adduced evidence that when the reaction of the blood swings toward the acid side reserve protein is transformed into "available protein," and this undergoes autolysis. They are of the opinion that a greater blood supply and a more complete removal of acid products may reverse the process and lead to a laying down of reserve protein and, therefore, to growth of the cell. This view is very suggestive that the tissues are quite susceptible to the medium in which their activities are carried on. It is quite plausible that a shifting in the point of equilibrium would bring about autolysis on the one hand and a synthesis of tissue on the other.

In the early phase of exercise the higher centers are utilized to great advantage, but later these changes are produced by chemical means. The hydrogen-ion not only acts upon the respiratory center but it also lessens the tone of the arterioles and capillaries and maintains an adequate supply of oxygen to the part. Furthermore, it accelerates the dissociation of oxyhemoglobin to the muscles and acts upon the vasomotor center.

Maladjustments of the cardio-respiratory mechanism produce interesting symptoms. Practically every symptom which occurs is explainable either upon a mechanic or neurogenic basis. One of the dominant features evidenced by ill-advised exercise is the rapidity of the heart, and frequently the patient becomes extremely cautious because of the palpitation. It is fair to assume in any chronic disease in which there is wasting of musculature that the nutritive condition of the heart must also suffer. In many cases of tuberculosis the heart has undergone a physiologic atrophy, due to

[1] Studies on Autolysis, III. The Effect of Reaction on Liver Autolysis, Jour. Biol. Chem., **25**, 261.

a lessened venous inflow. Primarily this is caused by costal inspiration, which lowers arterial pressure. As a sequel to the above we would expect a loss of contractile power of the heart, and such does occur, due to ventricular thinning. When exercise is instituted in these individuals the mechanical pumping action of the peripheral muscles squeeze the blood from the smaller vessels into the great veins, raising venous pressure and increasing the diastolic volume of the heart. The heart now dilates to its physiologic limits, but with some loss of contractile power it is unable to empty itself at the end of systole. To maintain the minute volume it is necessary that compensation occur, and this is effected by an acceleration of the pulse-rate or palpitation.

The early coördinating mechanism of exercise sends impulses via the descending tracts in the cord to the stellate ganglion, thus activating cardiac musculature and also to the vagal center producing a lessened tone. There are many cases, especially the incipient type, in which the palpitation is purely neurogenic. Furthermore, it is well to bear in mind the effects of costal inspiration and its relation to arterial pressure. It is not an uncommon occurrence in our cases for the pressure to drop below 100 mm. Hg and frequently below 90 mm. Markwalder and Starling[2] have shown that the coronary circulation is dependent upon the tone of the coronary vessels, but to a greater extent upon arterial pressure, and in the event that the pressure falls below 90 mm. Hg the blood supply is inadequate for the nutrition of the heart. The type of case which is most liable to show this is one in which fibrosis is extensive, and there is an associated pleuritis from apices to base. The heart being unable to adjust itself to this environment will attempt compensation and dilatation follows, which is the closing act in practically all forms of pulmonary tuberculosis.

Rise in body temperature during exercise is dependent upon the severity of the exercise. It is not uncommon to record a physiologic rise of 102°F. Loss of heat is dependent upon the outside temperature, humidity, presence or absence of winds and the nature of clothing worn. Dissipation is hastened in the patient who is lean and sparse, but a great deal slower in one possessing a large amount of subcutaneous tissue. Rise in the temperature of the body heightens the excitability of the nervous system and hastens metabolism. Physiologic rise involves carbohydrate and fat oxidation while a pathologic elevation produces protein destruction. Recent metabolic findings have shown that protein oxidation is not as great as we were formerly taught.

As impulses leave the cerebral cortex there is a wide distribution both supra- and infrasegmentally, and while a great many spend their momentum upon volitional control, an equal number activate

[2] A Note on Some Factors Which Determine the Blood Flow Through the Coronary Circulation, Jour. Physiol., **47**, 275.

those structures supplied by the vegetative system. During this dissemination stimuli are sent to the central sympathetic apparatus (Edinger) located in the midbrain and then migrate down the lateral splanchnic column, stimulating motor cells of sympathetic division and thereby producing a peripheral vasoconstriction and lessened dissipation of heat. Any impulse reaching the tuber cinereum, corpus striatum or any of the subsidiary thermogenic centers would produce a like result.

Those endocrine structures receiving a similar nerve supply become activated to a greater extent, and this probably accounts for the increased metabolism. Furthermore, the destruction of any foreign protein paraenterally shows a predilection for this particular division of the nervous system. Temperature should be considered as a beneficial phenomenon, and is simply the war that is being waged between the tubercle bacilli and the specific proteolytic enzymes generated by the tissues, and only by this procedure can organisms be annihilated. No longer should it be considered as a distinct entity, and we should bear in mind the numerous extraneous factors which may either cause elevation or depression, and remembering that toxemia is only one cause which provokes pyrexia. There are a great number of individuals who are distinctly sympathicotonic, and temperature in their respective cases is of least import.

When the activity within the lung has become quiescent the patient is then allowed to sit up, beginning with ten minutes each day and increasing ten minutes until they are sitting up either three or four hours. Exercise is then instituted, and the type most frequently resorted to is walking. Why should walking be given the preference over other forms of exercise? We unconsciously attempt to prescribe some form of exercise which is the most economic from the viewpoint of nerve expenditure. Every individual is trained in walking, and by utilizing this fact they receive the optimal benefit, and at the same time we are conserving the nervous system. Useless and ineffective movements are eliminated when we prescribe an exercise with which the patient is accustomed.

Every case should be individualized relative to exercise and never force walking to the point of fatigue. In the event that the patients complain of tiring no additional exercise should be indulged in until they are able to carry out their prescribed distance with comfort. The causation of the sensation of fatigue is rather obscure. Mosso long ago stated that "nervous fatigue is the preponderating phenomenon and muscular farigue also is at the bottom an exhaustion of the nervous system." There appear, however, to be two types of fatigue: one arising entirely within the central nervous system and the other in which fatigue of the muscles is superadded to that of the nervous system.

It has been suggested that fatigue may be produced by exhaustion

of the store of energy within the muscle and the clogging of the muscular machine by the metabolic products. During exercise the steady outflow of impulses to the muscles, the complex neural processes involved in any voluntary movement and the focussing of the attention upon the exercise must involve considerable expenditure. Hence it is easy to see that fatigue could be induced with very little exercise unless the walking is carefully gauged.

I have attempted to visualize exercise from a different phase than it is usually attacked and to appreciate the significance of the various symptom-complexes which may be produced in consequence of a pathologic process affecting the lungs.

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## POSTOPERATIVE MASSIVE COLLAPSE OF THE LUNGS.

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Rather singularly the subject of massive collapse of the lungs as a postoperative complication has scarcely been mentioned in American medical literature up to the present time, and only by English or Canadian authors. The importance of its consideration is accentuated by the fact that it undoubtedly occurs very commonly both in civil and military practice, and also because it is frequently confused with other more or less common postoperative pulmonary complications such as pneumonia, pleuritis, pleural effusions, etc.

Attention was first drawn to the occurrence of massive collapse of the lungs by W. Pasteur,[1] who cited 34 cases as occurring with postdiphtheritic paralysis of the diaphragm or other accessory respiratory muscles in 1890. It is interesting to note that Pearson-Irvine, in 1876, made the observation on cases which undoubtedly were cases of massive collapse, "That the physical changes observed in the lungs were the result of paralysis of the muscles concerned in the elevation and expansion of those parts." In a later series of 64 cases of postdiphtheritic phrenic paralysis, with 15 fatal results and with autopsies on 8 of these, Pasteur was able to demonstrate the gross pathology in 5, the others proving to be cases of bronchopneumonia. The cases were all bilateral, in a more or less advanced collapse, and all presenting the same characteristics *en grosse*, the parts being entirely devoid of air, of a deep, definitely circumscribed blue color and sinking entirely in water. Pasteur noticed the similarity in the symptomatology between numerous clinical cases developing postoperatively and these cases of postdiphtheritic paralysis

[1] Internat. Jour. Med. Sc., 1890.